some confidence that our correction is valid.

Using this correction, we calculated the percentage of the increment in respiratory evaporation above that observed at 20°C which could be accounted for by the volume of fluid secreted by both lateral nasal glands. The percentages were 33 at 31°C, 19 at 40°C, and 36 at 50°C. The differences between these figures are not significant, and we conclude that the lateral nasal gland accounts for about 30 percent of the increment.

The fluid secreted by the lateral nasal glands was hypoosmotic to plasma and contained less than 0.1 mg of protein per 100 cm^3 (*8*). There was a slight increase in Na^+ concentration and osmolarity with increasing rate of secretion; however, this was not statistically significant in our experiments. The rates of secretion and the compositions of the fluid secreted at various temperatures were: 2.6 g (gland · hr)$^{-1}$, 18.7 meq Na^+ per liter, 20.7 meq K^+ per liter, 75.7 milliosmols per liter, at 30°C; 4.5 g (gland · hr)$^{-1}$, 21.4 meq Na^+ per liter, 20.1 meq K^+ per liter, 80.7 milliosmols per liter, at 40°C; and 9.6 g (gland · hr)$^{-1}$, 35.3 meq Na^+ per liter, 18.6 meq K^+ per liter, 103.3 milliosmols per liter, at 50°C.

We conclude that (i) the rate of secretion from the lateral nasal gland increases with increasing respiratory evaporation as a result of thermal panting; (ii) between 20 and 40 percent of the increment in respiratory evaporation at air temperatures between 30° and 50°C can be accounted for by this secretion; and (iii) the location of the orifice of the single duct anterior to the turbinates may be essential to avoid desiccation of nasal mucosa during thermal panting.

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4. The orifice of the duct lies halfway between the rostral extremity of the dorsal nasal concha and the orifice of the nasal lacrimal duct.
5. We thank Howard Evans of Cornell University for providing us with anatomical preparations and injections of the gland.
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7. Air was metered through the mask at a rate of 60 to 150 liter/min STP. A sample of this air was metered through drying tubes immersed in an alcohol bath at −70°C. All flow meters were calibrated to better than ±1 percent accuracy with a Brooks Vol-u-meter. A blank value was obtained from a stream of room air drawn simultaneously through drying tubes at identical rates of flow. The amount of water frozen in the drying tubes was determined by weighing on a Mettler balance (model H-10). To determine the accuracy of this method, water was evaporated into the mask at known rates equivalent to those encountered experimentally. Recovery was between 98.7 and 101.8 percent.
8. Osmolarity was measured with an Advanced Osmometer (model 65-31), sodium and potassium were measured with an Instrumentation Lab flame photometer (model 343), and protein was measured by the method of R. J. Henry, C. Sobel, and S. Berkman [*Anal. Chem.* **29**, 1491 (1957)].
9. This work was supported by grant GB 27539 from the National Science Foundation.

24 April 1972

Chromosome Banding Patterns in Preimplantation Mouse Embryos

Abstract. *The chromosomes of first cleavage mouse embryos show the same banding patterns after quinacrine mustard staining as do chromosomes of differentiated mouse cells. Whatever feature of chromosome structure the banding pattern reveals thus appears not to be altered during development and differentiation.*

No tissue-specific differences in the banding patterns displayed by chromosomes after quinacrine mustard (QM) staining have yet been demonstrated. Mouse fibroblast chromosomes (*1*) show the same patterns as chromosomes of mouse bone marrow cells (*2*), and the chromosomes of human peripheral leukocytes (*3*) show the same patterns as do those of human primary spermatocytes (*4*). This is not unexpected if the QM bands reflect some feature of DNA composition (*5*), but they may reflect instead the distribution of a protein or proteins along the chromosomes. Thus the same bands as seen after QM staining can be induced by treatments known to be active toward protein rather than DNA, such as trypsinization (*6*) and treatment with sodium dodecyl sulfate, mercaptoethanol, and urea (*7*). Also it is possible that the acetic-saline-Giemsa technique, widely assumed to involve denaturation and renaturation of chromosomal DNA (*8*), could produce banding by selective protein elution from the chromosomes. If the bands reflect the distribution or types of proteins bound to DNA, or both these factors, then developmental alterations in the banding patterns are at least a formal possibility. We therefore

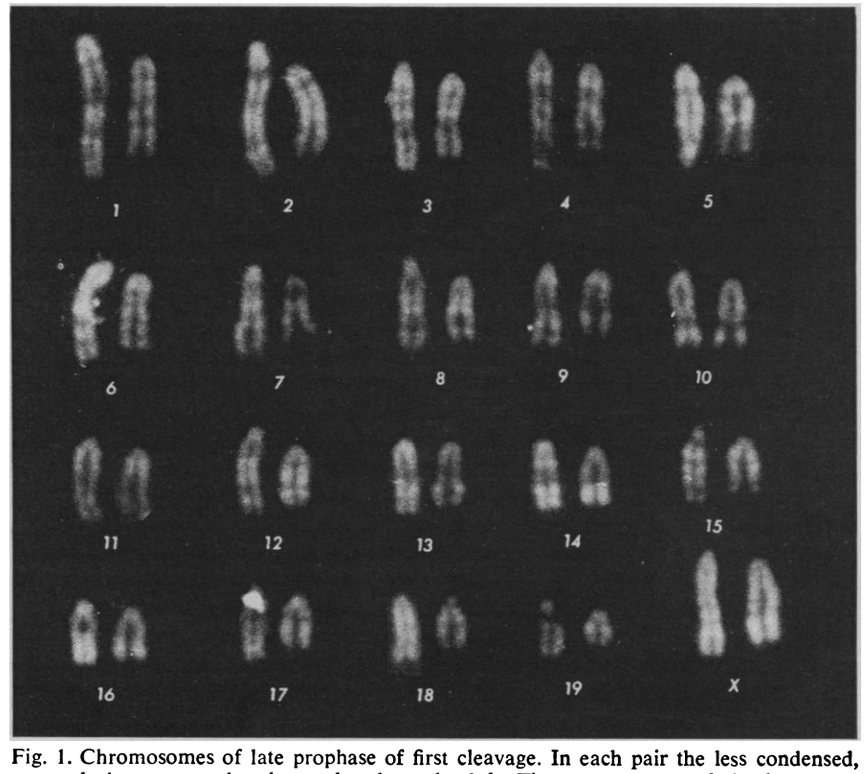

Fig. 1. Chromosomes of late prophase of first cleavage. In each pair the less condensed, paternal chromosome has been placed on the left. The arrangement of the karyotype is according to the recommendation of the Committee on Standardized Genetic Nomenclature for Mice (*12*).

thought it of interest to compare the chromosome banding patterns of early mouse embryos with those of differentiated mouse cells.

Female mice (*Mus musculus*) (CF-1; Carworth, Inc.) were superovulated and placed with CF-1 males when human chorionic gonadotrophin (HCG) was given. Mated females were injected with 50 μg of colchicine; the interval between HCG and colchicine administration was 28 hours for first cleavage embryos, 52 hours for second cleavage embryos, 72 hours for eight-cell embryos, and 100 hours for blastocysts. Four to five hours after injection of colchicine, embryos were flushed from the fallopian tubes or uteri, exposed to 1 percent sodium citrate, and fixed in methanol : acetic acid (3 : 1) (*9*). Slides were than stained with QM (100 μg/ml), and suitable chromosome spreads were located and photographed with the fluorescence microscope (*1*).

Figure 1 shows a karyotype prepared from an embryo at late prophase of the first cleavage division, when the maternally and paternally derived chromosome groups are still separate (*10*). The less condensed paternal chromosomes exhibit the same banding patterns as do the more contracted maternal ones. We also observed five other complete and several incomplete cells at first cleavage, about ten incomplete cells from second cleavage embryos, five complete cells from eight-cell embryos, and eleven complete cells from blastocysts. In all these embryonic stages the banding patterns are the same as those found in mouse fibroblasts and bone marrow cells (*1, 2*).

It has been suggested (*11*) that regions of chromosomes which fluoresce relatively brightly after QM staining are in general the same as those regions which replicate relatively late. Since late replication is a property generally attributed to heterochromatin, this suggests in turn that the brightly fluorescent regions contain heterochromatin. However, the fluorescent pattern of the facultative heterochromatin of the inactive X chromosome in mammalian females is the same as that exhibited by the euchromatic, active X (*3*). Insofar as the bright-staining regions can be taken to represent constitutive heterochromatin (*11*), our results indicate that its chromosomal distribution is established by the time chromosomes are first seen in the embryo, about 16 hours after fertilization. Our results therefore add data on the earliest embryonic stages to the accumulating evidence that the feature of chromosome structure which is revealed by QM staining does not change during development or differentiation.

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14 June 1972

Vampire Bat Control by Systemic Treatment of Livestock with an Anticoagulant

Abstract. *The blood of beef cattle given single doses (1 milligram per kilogram of body weight) of diphenadione (2-diphenylacetyl-1,3-indandione) became toxic to vampire bats (*Desmodus rotundus*) and remained toxic for 3 days without harming the cattle. Cattle at three ranches in Mexico treated with single intraruminal injections of diphenadione experienced a reduction in vampire bat bites of 93 percent. Bioassays of milk and liver from cattle treated orally with diphenadione in the laboratory indicated that there were no residue problems.*

We have developed a method of reducing populations of vampire bats that attack cattle by treating cattle systemically with a single intraruminal dose (1 mg/kg) of an anticoagulant, diphenadione (2-diphenylacetyl-1,3-indandione) (*1*). Vampire bats (Desmodontidae) feed exclusively on the blood of live vertebrates. The family's three genera, particularly *Desmodus*, the most abundant, are important carriers of paralytic rabies, the most serious animal health problem in Latin America; losses attributable to rabies, estimated at a million head of cattle a year, plus malnutrition, myiasis, and loss of blood resulting from vampire bat attacks, cost the Latin American cattle industry up to $250 million annually (*2*).

Mass vaccination of cattle has been the principal weapon for rabies control (*2*). Before 1970, no specific method for reducing vampire bat populations was known (*3*), even though control efforts with gassing, poisoning, shooting, netting, trapping, and dynamiting date back to 1934 (*2*). In 1968, the U.S. Bureau of Sport Fisheries and Wildlife, under an agreement with the U.S. Agency for International Development and the government of Mexico, initiated research to develop effective, economical, and safe methods for reducing the attacks of vampire bats on livestock through population reduction. One technique developed in this program has been described by Linhart *et al.* (*4*). Bats are mist-netted near cattle, treated topically with petroleum jelly containing a slow-acting toxicant, and released; when treated bats return to the roost, the toxic jelly is spread to other members of the colony and is ingested in grooming. The second method, described here, capitalizes on the daily requirements of vampire bats for blood, the tendency of anticoagulants to temporarily bind to the blood protein, and the differential sensitivity of cattle and vampire bats to diphenadione. In laboratory studies with caged (unexercised) common vampire bats (*Desmodus rotundus*) given diphenadione, we found that the single oral LD_{50} (dose statistically estimated as lethal to 50 percent of the population) is 0.91 mg/kg, but that a single oral dose of up to 5 mg/kg in cattle produces no observable signs of intoxication beyond a moderate increase in the clotting time of plasma prothrombin (the clotting time of whole blood was essentially unaffected). In one experiment, as little as 1 mg/kg of diphenadione was injected into the rumen of three beef calves (6 to 9 months old), and blood samples were taken at 24-hour intervals after treat-